disease. Treatment today offers a much better prognosis in mucoviscidosis than formerly,[1] and the possibility of this condition should be borne in mind in all infants with bacterial pneumonia.

The most important requirement in the management of infants with severe respiratory infections is adequate oxygen therapy. This aspect is well discussed in the article by H. Simpson and D. C. Flenley,[2] to which Dr. Gardner and his colleagues refer. Simpson and Flenley stress the importance of cyanosis in assessing severity. They also point out that the risk of inducing carbon dioxide narcosis in young children requiring oxygen therapy is apparently negligible. Inspiratory oxygen concentrations will frequently need to be considerably in excess of 40%, a figure which in practice is often not achieved in oxygen tents now available.[3] Severe hypoxia may result if the oxygen concentration falls suddenly,[4] and care is therefore essential when attending to children who are being nursed at high concentrations of oxygen. As in the adult, repeated blood gas analyses must be undertaken for the correct management of severe degrees of respiratory insufficiency. Thus Simpson and Flenley suggest that a pH below 7.2 or Pco_2 above 65 mm. Hg might be suitable indications for giving parenteral bicarbonate or intermittent positive pressure respiration or both. Though the value of tracheostomy in bronchiolitis has been questioned,[5] some children have been shown to benefit.[6] Complications are common after this procedure, however,[7] and nasotracheal intubation appears to be a preferable alternative.[8] [9] This too is not entirely without its problems, and it appears that it is preferably restricted to children with lower respiratory diseases.[10] These aspects of management are highly specialized and will be possible only in hospitals with facilities for intensive care, the requirements for which were discussed by Drs. D. Campbell, J. M. Reid, A. B. M. Telfer, and W. Fitch in the B.M.J. last week.[11]

The place of antibiotics in bronchiolitis was discussed in these columns two years ago.[12] As it is a viral infection, they will be of value only in preventing or treating the rare[13] but disastrous secondary infection. The correct choice may be difficult, but it is necessary to recognize that infants who were born in hospital, and especially those who have already received broad-spectrum antibiotics, are likely to harbour organisms highly resistant to antibiotics. No time should therefore be lost in testing bacterial sensitivities in any infant thought to have pneumonia, and therapy should include a penicillinase-resistant antibiotic such as cloxacillin. Tetracycline or chloramphenicol are suitable antibiotics for use in H. influenzae epiglottitis, and chlortetracycline is a useful prophylactic for an unprotected infant who comes into contact with pertussis.

Other aspects of therapy which may play a part in these severe diseases of early childhood include attention to fluid and electrolyte balance, mist therapy,[14] and correct position for nursing. Cautious sedation with, for example, chloral hydrate is often indicated for the restless infant with bronchiolitis after hypoxaemia has been corrected. The value of corticosteroids in bronchiolitis is doubtful,[15] but the occasional severely ill infant appears to derive benefit.

Cowpox and Paravaccinia

People whose daily work brings them into contact with farm animals are especially liable to develop bacterial infection such as brucellosis or rickettsial infection such as Q fever. It is less well known that they are also exposed to the risk of viral infections of the skin which may be acquired from cattle or sheep. The viruses concerned belong to the large group of poxviruses. The most important poxvirus in human disease is variola or smallpox virus, but this does not affect any animal species other than man. However, man is susceptible to other poxviruses whose natural hosts are cows and sheep.

Jenner's classic treatise in 1798[1] contained the first description of human cowpox, which at that time was not uncommon among dairymaids. Cowpox causes vesicles on the teats and udders of cows, and they progress to pustules, with the formation of scabs. It is a mild infection in that animal, and the disease is spread mainly by the process of milking. Cases of human cowpox are rarely reported, but A. W. Downie[2] found 10 outbreaks in cattle in England and Wales during 1944 to 1950, and in each instance attention was drawn to the disease by cases of infection in milkers. In 1909 R. J. Reece investigated an outbreak in Somerset which involved 23 out of 44 milkers and 214 out of 410 cows.[3] More recently W. F. T. McMath and H. T. H. Wilson described a case in this journal[4] of a young man who developed multiple lesions on the chin and lips from contact with an apparently healthy calf.

Human cowpox usually affects the hands—often between the thumb and forefinger—but lesions are sometimes seen on the forearm and face. The lesions are vesicular and closely resemble the reactions seen after successful vaccination against smallpox. Cowpox vesicles tend to be more haemorrhagic than vaccination reactions, and the vesicle fluid may be blood-stained. The lesions are painful and are usually associated with lymphangitis and lymphadenitis. There is often a mild constitutional upset, with some fever. The lesions are slow to heal and may take several weeks to resolve. The vaccinia virus used for vaccination against smallpox is very similar to cowpox virus and probably originally derived from it. Both have the brick-shaped particles on electron-microscopy which are seen with smallpox virus also. Cowpox and vaccinia have a similar range of susceptible hosts, and there have been outbreaks of cowpox in cattle due to infection introduced by a recently vaccinated person.[5]

In addition to cowpox, cattle are susceptible to other poxviruses that can infect human skin. These are members of the paravaccinia subgroup of poxviruses and include the virus of milker's nodules, bovine papular stomatitis virus, and the virus of orf or contagious pustular dermatitis in sheep.[6] Paravaccinia viruses have oval particles on electron-microscopy with characteristic criss-cross surface banding, so that they resemble coiled skeins of wool.[7] The virus of milker's nodules affects the teats and udders of cows, causing lesions

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[4] Campbell, E. J. M., ibid., 1965, 1, 1451.
[5] Wright, F. H., and Beem, M. O., Pediatrics, 1965, 35, 334.
[6] Canby, J. P., and Redd, H. J., ibid., 1965, 36, 406.
[7] McClelland, R. M. A., Brit. med. J., 1965, 2, 567.
[8] McDonald, I. H., and Stocks, J. G., Brit. J. Anaesth., 1965, 37, 161.
[9] Rees, G. J., and Owen-Thomas, J. B., ibid., 1966, 38, 901.
[10] Downes, J. J., Striker, T. W., and Stool, S., New Engl. J. Med., 1966, 274, 226.
[11] Campbell, D., Reid, J. M., Telfer, A. B. M., and Fitch, W., Brit. med. J., 1967, 4, 255.
[12] Brit. med. J., 1965, 2, 714.
[13] Disney, M. E., Sandiford, B. R., Cragg, J., and Wolff, J., Brit. med. J., 1960, 1, 1407.
[14] Avery, M. E., Galina, M., and Nachman, R., Pediatrics, 1967, 39, 160.
[15] Dabbous, I. A., Tkachyk, J. S., and Stamm, S. J., ibid., 1966, 37, 477.

which clinically bear some resemblance to those of cowpox. Orf produces lesions in the mouths of sheep and is mainly seen as an infection of lambs. The third paravaccinia virus—bovine papular stomatitis virus—is now thought to be the same virus as that causing milker's nodules.[8] Bovine papular stomatitis has recently been reported in Britain[8][9] and causes lesions which are clinically similar to milker's nodules on the hands and forearms of human handlers.[8][10][11]

Human infection with the paravaccinia viruses of orf and milker's nodules is not uncommon in rural areas. Infection is seen in people who have contact with cows or sheep or with products derived from them. For example, orf is seen in shepherds, butchers, sheep shearers, and veterinary surgeons and has been described in housewives with a liking for sheep's heads.[12-19] Milker's nodules are also seen in veterinary surgeons and in farm workers who tend dairy cattle—even if the animals are milked by machine.[5][20][21] Both viruses cause similar disease in man, and the name ascribed to the infection depends on the animals which were the probable source of infection.[20] As a result, orf is diagnosed in shepherds and milker's nodules in cowmen. Clinically the lesions are usually single, but cases with two or with multiple lesions have been reported. The fingers and hands are the commonest sites for them, but lesions have been described on the arms, face, neck, and occasionally on the leg. Different types of lesion may be seen. Most start as bluish-red papules or nodules, which are sometimes surmounted by a small white blister. Ulceration is common. Lesions may also take the form of granulomas in which there is ulceration with proliferation of grey-coloured epidermal tissue. Many appear to be vesicular, but it is often difficult to extract fluid from them.[22] The most striking feature is that the lesions are painless, though they look like acute inflammatory reactions. There is usually no constitutional disturbance. Lymphangitis and lymphadenitis have been described, but—in contrast to human cowpox—appear to be rare. The lesions persist for about five to eight weeks and resolve spontaneously without treatment. One attack appears to confer long-lasting immunity against reinfection, but, unlike cowpox, there is no cross-immunity with vaccinia.[22][23]

The relationship between the three paravaccinia viruses is obscure, though it is clear that they are closely related. J. Nagington and his co-workers have shown that orf and

milker's nodules viruses are similar in their morphology and cultural characteristics.[20] In addition I. M. Lauder and his colleagues have shown that the bovine virus of milker's nodules can produce lesions in lambs after inoculation on to the gums and lips.[21]

Neither cowpox nor paravaccinia is a serious infection in man. Diagnosis will present few problems to general practitioners in rural areas or to dermatologists, who also see these diseases from time to time. However, the infections might well puzzle a newly qualified casualty officer in a city, who may come across lesions on the hands of butchers, slaughtermen, or veterinary surgeons. When the infection has been diagnosed, the patient can be reassured—firstly as to the nature of the lesion, and secondly that it will resolve spontaneously and without complications within the following month or two.

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Pseudomonas Infection in Hospital

In the last few years the problems of staphylococcal cross-infection seem to have become more manageable, thanks especially to the semisynthetic penicillins tolerant of penicillinase. Unfortunately the same cannot be said of infection with Pseudomonas aeruginosa (pyocyanea). Indeed, reports of such infections have become more frequent. This can be partly explained by the relative ineffectiveness of chemotherapy for pseudomonas infection. Successful treatment of infections with Gram-positive cocci has undoubtedly brought the less tractable infections with Gram-negative bacilli such as Ps. aeruginosa into prominence. A relative and absolute increase in the amount of infection by these opportunists appears to have occurred.[1-3]

Epidemiological features of pseudomonas infection which have made it hard to eradicate[4] include its tendency to grow or at least survive in moist environments and its relative insensitivity to certain disinfectants as well as to most antibiotics. Moreover, despite a low pathogenicity for healthy people, it tends to be more pathogenic and invasive than most other common bacteria in patients or in tissues with low humoral resistance—for example, in infants and in leukaemic patients, in the chambers of the eye, and on the meninges. Not surprisingly, Ps. aeruginosa is a special hazard to patients treated with corticosteroids and with immunosuppressive drugs.[3-5] In burned patients two factors encourage pseudomonas infection—the presence of moist slough, which is easily colonized by Ps. aeruginosa, and (when the burns are extensive) reduced resistance to invasion.

The sources of these infections are varied and often elusive. Unlike Escherichia coli and Staphylococcus aureus, Ps. aeruginosa is usually not carried in large numbers by healthy people. Recent studies[6] have shown a larger proportion (about 12%) of healthy people to be carrying Ps. aeruginosa, at least in small numbers, than most previous surveys had suggested, and in another recent report[7] about 5% of healthy people were found to carry Ps. aeruginosa in the saliva. But, while sporadic self-infection with Ps. aeruginosa can undoubtedly occur, most endemic infection such as in burns and all outbreaks can be attributed to contamination from another human source or the inanimate environment. In many cases the source has been traced by typing of strains.[6][8-10] Human sources are generally sites of heavy infection—wounds, burns, the urinary tract. Established inanimate